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Literature review on canine parvoviral enteritis variants in Nigeria

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Introduction

Canine Parvoviral Enteritis [CPE] is acute highly contagious and often fatal viral enteritis of domesticated and wild canidae [dogs, foxes coyotes]. Caused by three variants of a virulent single stranded naked DNA virus belonging to the family Parvoviridae designated Canine Parvovirus Type 2 [CPV-2]. This disease is transmitted to susceptible animals directly via inhalation of viral particles or by ingestion of food contaminated by infected feces excreted by sick puppy or indirectly through contact with contaminated formites by infected feces excreted by clinically sick animal. The virus has high tropism for rapidly dividing cells, it is similar genetically to Feline Panleucopenia Virus [FPV]. Infact it only differ from FPV by two amino acids in it viral capsids. It was first reported in 1978 in North America and it became pandemic within six months of it first official report has two major clinical manifestation

Keywords: canine parvovirus, canine parvoviral enteritis, antigen, antigenicity, mutagenicity, feline panleucopenia, viral capsids

Abbreviations: CPE, canine parvoviral enteritis; CPV, canine parvovirus; CPV-1, canine parvovirus type 1; CPV–2, canine parvovirus type 2; FPV, feline panleucopenia virus

Myocardiac form

Enteric form

although this disease is still pandemic and has high morbidity and mortality rate in susceptible and immune compromised puppies and whelping bitches, it is incidences has been reduce greatly through appropriate vaccination protocol. Despite availability of commercially available vaccines, sometimes we still have documented evidences of CPE occurring in previously vaccinated puppies.

Literature review

Canine Parvoviral Enteritis is acute, highly contagious and often fatal viral enteritis of young puppies between the ages of six weeks to six months and immune-compromised whelping bitches caused by Canine Parvoviral Type-2 variants [CPV-2]. It was designated Canine Parvoviral Type 2 because another virus known as Canine Parvoviral Type 1[CPV-1] or Minute Canine Parvovirus has been isolated and identify previously in 1967 but this new viral isolates was found to cause a more fatal diseases in young puppies and it is antigenically different from CPV-1 viral isolates which are not as pathogenic and virulent as CPV can be found in feces of apparently normal dogs.

CPV-2 is very virulent and more pathogenic than CPV-1 cause more ftal disease and share more antigenic similarities with Feline Panleucopenia Virus [FPV] and Mink Parvovirus Enteritis than with CPV-1, in fact CPV-2 differs from [FPV] by two amino acids in it viral capsid. CPV-2 was first officially recognized as the cause of highly contagious new endemic fatal dog disease in North America in 1978 and later in Japan, Europe and Australia but subsequent retrospective serological studies of sick dogs serum indicates that this virus began infecting dogs in early 70s, this was due to finding of viral specific antibodies for CPV-2 found in stored serum of sicked dogs serum in Greece in 1974, Netherlands 1976, and Belgium in 1977. In 1978 serological studies of dogs serum carried out in dogs in Japan, New Zealand, Australia and United State of America confirmed the present of the virus in those countries. Canine Parvoviral Enteritis was first reported in Nigeria in Zaria in 1984 Adeyanju et al,[2] and later in southern part of the country in 1985.[1] This virus has strong affinity for rapidly dividing cells; it first replicates in lymphoid tissues of Oral cavity and pharynx, thymus, bone marrow mes enteric lymph nodes before it is disseminated into small intestinal crypts epithelial cells.

CPV-2 by infecting the lymphoid tissue causes suppression of puppy's immunity to infections and diseases directly through lyses of lymphocytes and indirectly through bone marrow depletion of lymphocytes progenitor stem cells inside the bone marrow. Viral replication in lymphoid tissues leads to marked atrophy of lymphoid tissues in thymus, spleenic follicles, lymph nodes of peyer patches, the same viral replication activities in epithelial cells of intestinal crypts lead to necrosis and sloughing off of intestinal lumen.

Virology of canine parvoviruses

There are two distinct canine parvoviruses.

a) Canine Minute virus or Canine Parvovirus type 1 Canine Parvovirus Enteritis or Canine Parvovirus type 2

Canine parvovirus type 1

This is non pathogenic Canine Adeno associated virus also known as Canine Parvovirus Type 1 or Canine Minute Virus; this virus has been isolated from feces of normal dog. It is widespread, it is not as pathogenic as CPV-2 and it is antigenically different from Canine Parvovirus type-2 but similar to Bovine parvovirus genetically, it was first isolated in military dog in1967 as the causes of mild diarrhea, due to subclinical enteritis, CPV-1 has causes pneumonia, myocarditis and lymphadenitis in puppies of 5 days to 21 days old. Most affected puppies has a mild disease but some might has a serious clinical form known as fading puppy syndrome CPV-1 causes infertility in bitch, stillbirth or abortion in pregnant bitch because of similarity with CPV-2 and CHV a thorough diagnostic work up is needed to confirmed CPV-1, use of PCR or immunoelectron microscopy are needed to diagnose CPV-1. At present no commercial vaccine is available for CPV-1, it is can only be prevented by maintaining cleaning environment in whelping bitch and avoiding overcrowding in shelter animal kennels.

Canine parvovirus type-2

Canine Parvovirus Type, also know simply as PARVO, it is highly pathogenic parvovirus that affect domesticated and wild canids it is a single stranded, non envelop DNA virus, that is extremely resistant to various disinfectant but susceptible too and easily destroy by sodium hypochloride, it is highly mutagenic and it is believe to still be in it evolving stage, it is believe to evolved from mutation from Feline Panleucopenia virus or other carnivore Parvoviruses because it differs from Feline Panleucopenia virus [FPV] and Mink Enteritis Virus by only few DNA bases in its viral capsid[3–5] this might probably close association of dog and cat kept together as companion animal or due to laboratory tissue culture contamination and it worldwide fast distribution by vaccine contamination, however these assumptions has not been proved.

Virus morphology

CPV-2 virion particles are small viral particles, spherical in shape approximately 20nm in diameter and none enveloped.[6,7] The CPV-2 was first isolated in1978 and by1980 it has become panzootic with new strains of the virus isolated in 1979 designated CPV2a, this new strain has replaced the original CPV-2 virus in most infected dog viral isolates and this new strain is found to be more infectious to cat than original CPV-2. There are only small antigenic variations between strains of CPV-2 [CPV-2a, CPV-2b, CPV-2c] detectable only by monoclonal antibodies and genetic analysis. CPV-2a was discovered in 1979[8,9] and by 1980 has replace the original CPV2 in circulation another new strain was discovered in 1984 designated CPV 2b this differ from CPV2a by one or two amino acid substitution in it viral capsids 11 [VP 2]. CPV 2a differs from original CPV-2 by having different amino acid within it viral capsid 2 [VP2] at the following viral capsid epitome positions Met87leu, Ile101Ther, Ala300Gly and Asp305Tyr different from amino acid found in original CPV- 2 at such virus epitome position.

Another viral strain was discover to have had another changes as position Asn 426 Asp[10] and position 297 with amino acid substitution of serine to alanine [Ser to Ala]this new strain was named CPV2b in 1984 and another strain was discover in Italy with distinctive antigenic characteristic and amino acid sequence changes at 426 with glutamine been substituted for Aspagine thus altering the viral antigenic site epitome A, CPV2c has only one amino acid altering at position 426 I.e GLU 426 [Asp 426 Glu]. These new strains have a wider host range than original CPV2 and can infect cat more readily than Feline Panleucopenia virus.[11]

Antigenicity

CPV-2 is closely related antigenically to FPV and MEV[12] but it has no antigenic similarity or relationship with Canine Minute Virus or CPV-1[13] or with Dependo virus associated Canine Adenovirus it shares minor serological cross reactivity reactions with Swine Parvovirus, CPV 2 affect all members of canidae. also it two clinical manifestation enteritis and myocarditis are diseases not previously seen in dog, serological survey shows that CPV-2 was a new viral infection,[14] the earliest known antibodies associated with CPV-2 is the one found in dog sera in Greece 1974 and Belgium 1976 CPV 2 has high rate of nucleotides substitution rate similar to RNA viruses CPV 2a and CPV2b are antigenically similar to original CPV 2 viral isolates even though there were some amino acid substitution in the amino acid sequence of their viral capsid protein CPV 2a show several amino acids substitution changes in it viral capsids amino acid sequence, that gave it distinct antigenic characteristic different from CPV2 at viral capsid position Met87Leu, Ile101Thr, Ala300Gly and Ala305 Tyr different from original CPV2.

CPV2b also contain these aforementioned changes plus one additional substitutional changes at viral capsid position 426 [Asn426Asp] another variants of CPV 2a Ile324 was found to be limited to Asian countries I.e. Thailand, Japan, China, India and Korea, this Asian strain of CPV2a has amino acid sequence substitution at position 324 which is adjacent to position323 viral capsid, this viral capsid epitome site is important in virus virulent characteristic and host range specificity together with viral capsid position 93, CPV viral capsid position 323 and 93 play an important role in host range specificity and tropism for canine transferrin receptor binding.[15] Another distinct strain was discover in Italy in year 2000 with distinct antigenic characteristic different from CPV 2a and CPV2b and slight variation in it viral capsid at position 426 known as Glu 426 mutant or CPV2c because glutamic acid was substituted for Asparagin/Aspartic acid at position 426, it is more virulent, spread more rapidly and can infect cat more readily than FPV,[11] Buonavoglia C,[10] Martella et al,[16] also similar and unique antigenic changes was found in a strain in China and Taiwan in CPV2c position370 Gln370 Arg, this changes were similar to the one found in China Panda parvovirus population.[17] Position 370 is adjacent to viral capsids site 359 and 375 which make it flexible unique surface loop of capsid protein, also viral capsid site 359 and 375 are adjacent to viral capsid double Ca^{++} binding site which is very important in determining viral infectivity. Any changes in these sites affect virus ability to heamagglutinate red blood cells[18] the changes in CPV2c occur at distinct and important antigenic determinant variation sites that make it to have a different antigenic properties from CPV2a and CPV2b therefore most commercial vaccines prepared with CPV2a and CPV2b antigenic strains might not confer immunity against infection with field challenge with CPV2c infection.

Mutagenic abilities of CPV-2

CPV-2 was first discovered in dog in North America and Europe in 1978 as a new viral disease suspected to have mutated from Feline Panleucopenia Virus or Mink Enteritis Virus by 1979 this virus has attained a pandemic status. shortly and it was reported worldwide in 1979 and by 1980 a new viral strain has evolve from it, designated CPV2a, has evolved from the initial viral strain of CPV 2 with few genetical re assortment of few of it viral capsid protein bases that changes it virus antigenic characteristic detectable only by deep genetic analysis and monoclonal antibodies tests. Further minor antigenic shift occur in new viral isolates in suspected outbreak in 1984 this new isolated strain was designated CPV2b.

The virus has high rate of adaption to adverse environmental conditions and high mutagenic potentials, these abilities helps in virus high rate of spreading, it has shown remarkable ability to survival in the environment under adverse condition couple with viral high rate of nucleotide substitution only comparably to RNA viruses, this abilities has help CPV2 to mutate into a new more virulent, more pathogenic, more resistant and more stable with increase host range infecting abilities. CPV 2 is believe to still be in it evolving stage, these abilities has continue to account for persistent parvovirus enteritis infection seen today.[19] we now has three dominant strains that are mutant of original CPV 2 that causes disease in dog worldwide designated CPV 2a, CPV 2b, CPV2c.

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Breed susceptibility

Although all breeds of dogs are susceptible but Rottweiler, Doberman pinscher, America pitbull terrier, English Springer spaniel and German sherpherd dogs are believed to has a higher risk of coming down with the disease than other breed of dog.

Epidemiology

Parvoviridae are small, non enveloped single stranded DNA viruses that are sometime species specific in causing disease in mammalian animals Canine Parvovirus belong to the family Parvoviridae, genus Parvovirus, these are small viruses with DNA genome of about 5000 amino acids/bases with a hair pin morphology. Using X-ray crystallography it viral capsid have been found to be sixty copies of combination amino acid making up it three viral capsids designated as VP1, VP2 and VP3 VP1 has full sequence with additional N terminal domain, VP2 account for 90% of the viral capsid and it is the major determinant of host range or specificity and pathogenicity it cleaves to VP3 using host protease enzyme. Parvoviruses has exceptional ability to evolve into a more stable, more virulent strain with increasing host range infecting ability, this has help greatly in their ability to persist in the environment couple with the fact that they can survival in the environment under unfavorable condition with large amount of viral particle shed in feces by infected dog billion of viral particles are excreted by infected dog, this active shedding can last up to 2weeks. This virus has affinity for rapidly dividing cells, this account for it tropism for lymphoid tissue, myocardium cells of puppies under three weeks, bone marrow and intestinal crypts epithelium cells of dogs. Appel et al,[14] since 1981 most countries of the world has report presence of CPV 2 in their dog population but the most dominant strains isolated in Nigeria using SNAP parvo antigen test are CPV 2a and CPV2b Dongonyaro et al, and dominant CPV 2 strains isolates from South Africa are CPV 2a and CPV2c. It is possible we have undocumented CPV 2c strain in Nigeria because of large number illegal importation exotic dog breed from South Africa to Nigeria without adequate and proper quarantine procedure. Most adult dogs are now resistant to this disease because they must have acquired immunity against it either by survival natural subclinical infection or through vaccination against it.

Most breeding bitches are now immune against CPV2 strains and can pass maternal antibodies to their neonate via colostrum or via the placenta in the uterus. This help greatly in reducing myocardium form of the disease that is prevalent in susceptible puppies below the age of three weeks as the neonate has active maternal immunity for the first weeks of live when infection with CPV 2 can result in myocarditis.[20] this make myocardium form of the disease to be rare occurrence, occuring only exclusively in pup of individual non immunized pet bitch that comes in contact with the disease at about the time of whelping, one way this can occur is when such bitches has dystocia and they are presented for cesarean section[21] although severe clinical enteritis disease occur in dog younger than six month of age, adult dog with insufficient immunity may be at risk of infection too, if they come in contact with the disease at any age.[22]

Predisposing factors

Breeds: Certains breeds of dog show high risk of coming down with CPV-2 diseases than others, reason for this increase infectivity within breed is unclear bit it has been suggested that Doberman pinscher and Rottweler breeds of dog has high risk of coming down with CPV 2 because both breed share common ancestor, they both have higher prevalence of Willebrands disease couple with the fact that Rottweiler breed are predispose to genetic immunodeficiency, also these breed are more popular and common than other breeds, inadequate vaccine protection due to owner not following strict vaccination protocol in general. Also America pit-bull terrier, Labrador retriever are also among dog breed with high risk of been susceptible to canine parvoviral enteritis [CPE].

Sex: intact male older than six month are more likely to come down with CPE than intact bitch.

Seasonal variability: CPE is more common in summer than in winter and in Nigeria there is high prevalence of the disease from January to August, it peak occurs at February to April but no CPE are recorded in September to December.

Age incidence: dog of any age can be infected but the incident of clinical disease is more in puppies of weaning age six weeks to six month of age, puppies younger than six weeks are protected by maternal antibodies. More adult dog are already immune due to vaccination or sero conversion immunity from sub clinical infection, after six weeks maternal antibodies concentration start dropping belong protective concentration in the serum, until about 20weeks when it deplete to such a low concentration that it cannot protect the puppy from any infection. CPE can affect stray unvaccinated adult dog up to one year of age.

Shelter animal: due to exposure to many animals in close proximity confinement, puppies from shelter animal adoption centre are more likely to come down with the disease.

Malnutrition: malnourished animal have low immunity and therefore have high risk of easily succumbing to any environmental challenge.

Poor sanitation: infected puppies can sheds infective viral particles for up to two weeks, these viral particle can survive in unsanitary favorable environment and remain infective up to eighteen month, so animal kept in poor sanitary environment were at high risk of coming down with the disease.

Transmission: CPE is transmitted directly by feco oral route and indirectly through contact with contaminated formites, during illness sick animal continue to shed massive amount of viral antigen in feces these virion particles can survival in the environment for long time and retain their capability to be infective even long after cessation of clinical signs of disease, ingestion of contaminated formites from environmental contamination play a major role in transmission of CPE,[21] one gram of contaminated feces from actively shedding acute infected puppy is sufficient to infect at least one million susceptible puppies by oral route.[12]

Incubation period: sign of enteric disease appear in 4-14 days after exposure to viral particle.

Pathogenesis: After infection by ingesting viral particles through feco oral route or through inhalation of viral particle from contaminated formites, viral replication begins in lymphoid tissues specifically lymphoid tissue of the oral cavity and pharynx, mesenteric lymph nodes and thymus and it is disseminated through hematogenous route to rapidly dividing cells of intestinal crypts epithelium cells, this last for three to five days after infection, marked viremia developed in the plasma and it is noticed up to five days after infection. After plasma viremia, the virus is found in many rapidly dividing epithelium cells for example epithelial lining of the tongue, esophagus, oral cavity,

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small intestinal crypts epithelium cells, bone marrow, spleen, thymus and various lymph nodes. The severity of the disease is determined by cells turnover rate at these epithelial cells, higher cells turnover rate in lymphoid tissues and intestinal crypts epithelium means higher viral replication rate and more destruction of cells at these sites and more tissue necrosis observed. During four to six weeks of age enterocytes of the intestinal crypts has higher mitotic index and higher cell dividing and replication rate, this is due to the fact that around this time there is change in puppy's diet due to weaning and change in intestinal micro flora, this make puppies more susceptible to infection around this time. Parvovirus infects the germinal epithelium of the intestinal crypts causing destruction of the epithelium and villous damage and collapsed thus leading to characteristic pathological lesion of shortened and atrophic villi of the intestines; this altered the absorptive properties of the intestines epithelium cells in the gastro intestinal tract.

There is extensive lymphocytolysis in the germinal center and cortex of thymus because of higher mitotic index in the thymus, this is responsible for the lymphopenia found infected puppy. Early lymphoid tissue infection with overt clinical signs accompany by temperature raise and lymphopenia initiate the disease in all cases of clinical manifestation, there after myocardium cells and intestinal crypts epithelial cells are affected. In neonatal puppies rapid myocytes replication occurs during the first 2 weeks of life,[23] while intestinal epithelium cells turnover rate is slow during this time[20] these situations reverse itself in the following weeks, when intestinal crypts epithelial cells start replicating actively at four weeks of age, cardiac growth continue as hypertrophy not as replication although DNA synthesis and nuclear kinesis continue until at least 8weeks of age.[23] infection of susceptible neonatal puppies any time as from four weeks of age result in enteritis However infection in susceptible bitch at various stages of pregnancy does not cause intra uterine infection in fetus Meunier et al,[11] also Parvoviruses infection does not cause stillbirth or affect conception rate, Parvoviruses infection does not have any effect on reproduction as it does not affect incident rate of stillbirth, average litter size does not increase or reduces in an experiment conducted on two thousand brooding bitches.[12]

Clinical forms

There are two major clinical form / manifestation of the disease that is;

a. Cardiac form

b. Enteric form

Cardiac form

Seen in young neonatal puppies less than three week of age and immuno compromised bitches, it manifest as sudden death in apparently normal puppy after exposure to sudden stress, excitement or exercise. Affected puppy gasp, mucous membrane become cyanotic with death occurring under two hour of initial clinical manifestation due to non suppurative myocarditis, mortality may be up to seventy percent in affected litter. Surviving puppy from infected litter are susceptible to heart disease later in life. By eight to twelve weeks of age surviving puppy show sign of acute heart failure [cardiomyopathy] which include dyspnoe, tachycardia, tachypnoe with ascites and hepatomegaly[21] sudden death is due to irregular heart beats and delay onset of chronic congestive heart failure. Most affected puppies were infected immediately after whelping but because most bitch are now immune to CPE through natural field challenge or through proper vaccination protocol, there is passive transfer of maternal antibodies to puppies thus this form of the disease is rare.[12]

Enteric form

Enteric form of CPE is the commonest form of the disease, CPE is the most commonest cause of viral enteritis in young puppies of six weeks to six months, the infection start as non specific gastrointestinal tract disturbances. Affected puppies were withdrawn, lethargy, vomiting, diarrhea and as the disease progresses the diarrhea become blood tinged or bloody diarrhea, foul smelling, intractable fluidy, these signs are not limited to Parvoviral enteritis induces diarrhea, animal become dehydrated, hypothermic due to diarrhea and vomiting, icterus and hemorrhagic diathesis [Disseminated intravascular coagulopathy] may develop terminally[Otto C M.et al 2000]. Secondary bacterial infection may lead to bacteremia and endotoxemia. Bacteremia and endotoxemia may lead to systemic inflammatory responses [SIR] Death is due to dehydration and elctrolyte inbalance, leucopenia further exerbate immune system with may lead to endotoxic shock and comma.

Diagnosis

Suspect parvoviral enteritis in young puppy of six weeks to six months with no history of proper vaccination record again CPV-2 and shows the following clinical signs, active animal suddenly withdrawn to it, stopping eating for about two to three days, vomiting, lethargy, diarrhea. Depression and fever, this clinical signs are not specific for CPE and cannot serve as confirmatory diagnosis. Use of commercially available fecal enzyme immunoassay test [ELISA] can be use to performed rapid confirmatory diagnosis of CPE on the clinic floor.

Laboratory confirmatory diagnosis of CPE can be made with haemagglutination of pig, cat and Rhesus monkey RBC at PH of 6.5 at 4^0C with viral antigen from sick puppy fecal extract. The specificity of haemagglutional is determined by titration of the sample in parallel presence of normal and immunized dog serum. Freely infected dog fecal sample contains many thousands of haemagglutinating units of viral antigens electron microscopy can be used to performed confirmatory diagnosis viral isolation and identification from suspected sick animal fecal sample viral amplification of viral DNA using PCR assay of suspected fecal sample serology can be used for retrospective confirmatory diagnosis of suspected case or use of IgM or IgG capture enzyme linked immunosorbent assay on a pair sera or use of probe based real[24] post mortem lesion and histopathology studies of these lesion can also aid in definitive diagnosis of CPE.[24] There are slide agglutination test and slide inhibition test can detest all strains and genotype of CPV are commercially available using porcine erythrocytes.[25]

Radiography

Contrast radio graphic image of the gastrointestinal tract can detest pathological lesion in the abdominal lumen, although these changes are not specific to enteritis caused by CPE but they can aid in arrival at definitive confirmatory diagnosis, radio graphic changes observed include fluid and thinning of intestinal mucosa lining coupled with low intestinal motility.

Ultrasonography

Ultrasound examination of abdomen can detest abdominal and peritoneal effusion and intussusceptions.

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Clinical pathology

Prominent histological examination finding of complete blood cells in CPE cases include leucopenia due to neutropenia and lymphopenia as a result of destruction of bone marrow precursor stem cells due to viral replication activities lymphopenia is due to depletion of stem cells destruction of lymphoid tissue parenchyma and lysis of lymphocytes. Leucopenia is so severe that leucocytes count could be as low as 500- 2000 leucocyte per microlitre or less, more leucocytes count or rebound neutrophilia is useful indicator of recovery in sick animal.

Haematocrit

It can be variable, not specific good indicator, it can be low due to intestinal hemorrhage or high due to dehydration from fluid loss as result of vomiting and diarrhea.

Serum chemistry

Serum chemistry is not a good specific indicator of CPE because result obtained can be seen in other enteritis cases. Noticeable hypokalemia is due to anorexia, vomiting and diarrhea, hypocalcemia is due to hypoalbuminemia with may be relative hypoalbuminemia or absolute hypoalbuminemia with might be due to reduction in plasma protein concentration due to intestinal hemorrhage or hemodilution dilution due to fluid re hydration therapy, there is noticeable increase in alpha 2 globuline concentration despise reduction in plasma protein this can be due to hepatic synthesis of acute phase protein [APP] stimulated by endogenous leukocytes mediator that are produced as a result of tissue damage and inflammatory process, production of acute phase protein lead to reduction in albumin synthesis, there is increase in Alkaline phosphatase and Alanine transaminase as a result of reduce oxygen concentration in the liver due to low circulating blood delivered to the liver or due to many circulating bacteria endotoxin as a result of compromised intestinal epithelial absorption capacity due to destruction og GIT lumen, PH can be acidic or alkaline depending or predominant ion loss due to vomiting or diarrhea, [vomiting lead to loss of hydrogen ion and chloride ion loss, while diarrhea ion loss depend on the origin of the diarrhea it is small intestine or large intestine] majority of CPE cases show metabolic acidosis due to excessive loss of bicarbonate ion[HCO_3], unlike in human total ionized magnesium concentration cannot be use as a good indicator good prognosis.

Serology

Determination of positive antibodies against CPV can be misleading because 95% of dog population now has sero conversion due to previous exposure to CPV in sub clinical infection in the environment or through vaccination so they will test positive. Specific serology test for IgM analysis by indirect fluorescent antibody [IFA] or Mecaptoethanol procedure provide more definitive serological evident of recent infection because IgM is only found in first week of clinical infection. Positive definitive confirmatory diagnosis of Canine Parvoviral Enteritis required demonstration of active secretion of viral antigen in feces which can be done on site by [ITE-parvotest, IDEX, Assure parvovirus symbiotic] all these are commercially available ELISA test, easily to conduct and give reliable positive result which indicate active fecal excretion however recent vaccination with attenuated live vaccine may give similar result too.

Management

Chances of survival for clinically infected puppies increases if such puppies are place on intense veterinary medical hospitalization and clinical abbe ration signs and symptoms are managed as soon as they are observed. Although CPE start with non specific enteritis clinical signs, CPE should be suspected in any young puppies of six weeks to 6months with or without proper vaccination history coming down with any signs of enteritis disease, the treatment should commence as soon as possible, infected puppies has been chances of survival if they were placed on appropriate par enteral fluid therapy to manage electrolytes imbalance and dehydration due to vomiting and diarrhea couple with bactericidal broad spectrum antibiotic par enteral injections.

Fluid therapy

One of the major noticeable clinical signs of CPE is intractable vomiting and projectile foul smelling diarrhea, these clinical signs cause rapid electrolytes in-balance and depletion of electrolyte ion distorting the animal acid based balance and normal body internal homeostasis. Replacement and maintenance loss body electrolytes are one the major cardinal point of successful CPE management.

Determination of appropriate crystalloids to use in replacement of loss electrolytes is very important because in CPE both metabolic acidosis and metabolic alkalosis can be observed, determination of body PH can be done or use of isotonic crystalloids like normal saline and lactated Ringer, Ringer lactate solution should be use with cautions in cases of CPE with severe metabolic acidosis or metabolic alkalosis or if there is any indication extensive hepatic damage that might effect lactate metabolism or when administering ceftriazone as systemic antibiotic in CPE management. Also any puppies with noticeable hypersensitive reaction to cereal product should not be given any crystalloid that contains dextrose, metronidazole infusion should not be given with lactate ringer infusion over a long period of time. Intravenous routes is most preferred route of fluid administration because severe dehydration impaired absorption of fluid from subcutaneous routes, intravenous routes also help to rapidly replace and correct electrolytes in balance in circulation in puppies with hypovolemic shock colloidal fluid can also be administered with 50% isotonic crystalloid fluid to help improve circulation oncotic pressure loss due to high protein loss pottassium chloride at dose rate of 20 mEq/l is administered with fluid to help correct hypokalemia normally observe in CPE. Fluid replaced at a dose rate of 40-60ml /kg body weight multiple by percentage deficit.

Antibiotic

Par enteral administration of broad spectrum antibiotic that is bactericidal is essential in CPE management because of disruption of intestinal mucosal integrity with adversely affect intestinal normal micro flora population aminoglycosides are very effective in well hydrated puppy. Cephalosporin is very good but concomittant administration of ceftrizone with lactate Ringers solution should be avoided to avoid calcium precipitation.

Antiemetics

anti-emetics are very important set of drug use in CPE management because of frequent vomiting, metoclopramide is a dopaminergic antagonist that block chemo receptor trigger zone and also has

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prokinetic effect on GIT very effect but strongly contraindicated in CPE puppies with accompany intussusception. Ondasetron and dolastron both serotonin receptornantagonist can also be use in case of freqent uncontrollable vomiting metoclopramide, ondansetron and dolasetron are antiemetic that can act centrally and peripherally to stop nausea and vomitting caused by central and peripheral stimulation of vomiting pathway.

Nutritional support

Introduction of bland enteral feeding early has improve chances of puppies survival and improve earlier restoration of mucosal integrity faster.[26,27]

Antiviral treatment

since CPE strains share similar antigenic characteristic with Feline Panleucopenia Virus, use of Feline Recombinant interferon [rFeIFN-w] has been recommended and has given a promising result in tested case involving ninety four dogs with naturally occurring CPE infection there are noticeable drastic improvement in clinical conditions and severity of disease, dogs are treated for three days at dose rate of 2.5 micro gram per kilogram body weight of rFeIFN-w intravenously for three days Osteltamivir a neuraminidase inhibitor has been use to successfully improve affected CPE puppies hematological parameter and body weight when administer for five days at dose rate of 2mg/kg orally. It does not seem to have any effect on reducing mortality rate in affected puppies. Also human recombinant granulocyte colony stimulating factor [G-CSF] has been employed in the past in management of CPE with no documented benefit on treat outcome on overall management of CPE. Equine endotoxin antiserum has been used in the past with significant impact on treatment outcome. Bactericidal permeability increasing protein [$rBPI_{21}$] has been used in the past and it does not reduce seem to have any effect on endotoxin concentration in the abdominal lumen but intestinal mucosa protestant coating agent like sucralfate and H_2 blocker can be use.

Pain therapy

Colic as a result of hemorrhagic enteritis and intestinal intussusception is a common sign in CPE use of analgesic like Butorphenolor Buprenorphine are beneficial or Hyoscine butylbromide [Buscopan] an anticholinergic drug is also very good in reduce abdominal pain observed in CPE.

Prevention

Canine Parvoviral Enteritis can be effectively prevented by following strict vaccination protocol in susceptible pet population by using polyvalent vaccines containing antigen for canine distemper, canine hepatitis, leptospirosis, CPE and canine parain fluenza. These vaccines contain modified live strains of CPE and can be administer at 6-8weeks, follow by first booster at 10- 12 weeks and second boosting shot can be given at 14-16 weeks old they repeat at 6 months to 12 months later. This schedule is endorsed by world small animal veterinary association. Most commercially available vaccines contains modified live vaccines that can be use to prevent infection in susceptible animal or protect already infected puppy, some of these vaccine can provide immunity cover that can last up to 5-7years. Any puppy that succumbs to infection after completing the initial vaccination protocol at 16 weeks should be re vaccination twice at four week interval. In shelter environment or overcrowding population puppy can start receiving CPE Vaccine at four week old and repeat after 3-4 weeks later good hygiene and strict bio security sanitary protocol are very important in limiting outbreak of CPE infection in susceptible population wash all contaminate hard surface and formite with disinfectant with sodium hypochloride is very effective in killing the virus.[28–34]

Control

a) Proper vaccination schedule

b) Intense hospitalization of sick dog

c) Good nutrition

d) Good hygiene

e) Reduce overcrowding

Conclusion

Canine Parvovirus Enteritis although very contagious and lethal viral enteritis can be prevented by vaccination and good hygiene, also if susceptible puppy succumb, hospitalization of such animal greatly improve their chance of survival.

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None.

Conflicts of interest

Author declares that there is no conflict of interest.

References

1. Adeyanju JB, Abdullahi SU, Abdullahi R, et al. Canine Parvoviral Enteritis in eleven suspected cases in Nigeria dog. *Vet Med (Auckl)*. 2016;11(7):91–100.
2. Kamolu PB. Canine Parvovirus infection in Nigeria. *Journal of small animal practice*. 1985;26(11):663–668.
3. Carmicheal LE. Immunization strategies in puppy, why failure? *Compend continues Edu Pract Vet*. 1983;5(12):1043–1052.
4. Tratschin JD, McMaster GK, Kronaer G. Canine Parvovirus Relationship to wild type and vaccine strain of Feline Panleucopenia Virus and Mink Enteritis Virus. *Journal of General Virology*. 1982;61(1):33–41.
5. JW Black, MA Holsher, HS Powell, et al. Parvoviral Enteritis and Panleucopenia in dog. *Vet Med Small Anim Clin*. 1979;74(1):47–50.
6. Carmicheal LE. Canine parvovirus type-2 an evolving pathogen of dog. *Anales des MedicineVeterinarie*. 1994;138(7):459–464.
7. Eugster AK, Nairai. Diarrheas in Puppies parvovirus like particles demonstrated in their feces. *South West Vet*. 1977;3:59–60.
8. Parrish CR, O Connel PH, Evermann JF, et al. Natural variation of Canine Parvoviru. *Science*. 1985;29(230):1046–1048.
9. Parrish CR, Have P, Foreyt WJ, et al. The global spread and replacement of Canine Parvovirus strains. *J Gen Virol*. 1988;69(5):1111–1116.
10. Decaro N, Desario C, Parisi A, et al. Genetic analysis of canine parvovirus type 2c. *Virology*. 2009;385(1):5–10.
11. CE Greene, Decaro N. *Infectious Disease of Dog and Cat*. USA: WB Saunders Philadelphia PA; 2012.
12. Meunier PC, Cooper BJ, Appel MJ, et al. Experimental viral myocarditis, parvoviral infection in neonatal pup. *Vet Pathol*. 1984;21(5):509–515.
13. Appel MJ, Scott FW, Carmicheal LE, Isolation and immunisation studies of Canine Parvo like virus from dog with hemorrhagic enteritis. *Vet Rec*. 1979;105(8):156–159.

14. M Appel, P Meunier, R Pollock. Canine Parvoviral Enteritis, A report to practitioners. *Canine practices*.1980;7:22–36.

15. Hoelzer K, Parrish CR. The emergence of parvovirus of carnivores. *Vet Res*. 2010;41(6):39.

16. Buonavogalia C, MartellaV, Pratelli A, et al. Evidence for Evolution of Canine Parvovirus type -2 in Italy. *Journal of General Virology*. 2001;82:3021–3025.

17. Guo L, Yang SL, Chen SJ, et al. Identification of Canine Parvovirus with Q370R point mutation in the VP2 gene from Giant panda [Ailuropoda melanoleuca]. *Virol J*. 2013;10:163.

18. Simpson AA, Chandrasekar V, Herbert B, et al. Host range and variability of calcium binding by surface loop in the capsid of canine and Feline parvoviruses. *J Mol Biol*. 2000;300(3):597–610.

19. Goddard, Leisewitz. Canine Parvovirus. *Vet clin small animal*. 2010;40:1041–1053.

20. Meunier PC, Cooper BJ, Appel MJ, et al. Pathogenesis of Canine Parvovirus Enteritis, sequential virus distribution and passive immunization studies. *Vet Pathol*. 1985;22(6):617–24.

21. Macartney L, McCandlish IAP, Thompson H, et al. Canine Parvoviral enteritis, clinical hematological and pathological features of experimental infection. *Vet Rec*. 1984;115(9):201–210.

22. Marcovich JE, Stucker KM, Carr AH, et al. Effect of Canine Parvovirus strain variation on diagnostic test results and clinical management of enteritis in dog. *J Am Vet Med Assoc*. 2012;241(1):66–72.

23. Bishop SP, Hines P. Cardiac Muscle cytoplasmic and nuclear development during canine neonatal growth. In; RE Roy, P Harrus, editors. Recent advance studies in Cardiac structure and metabolism. Baltimore MD: University Park Press; 1975.

24. Yilmaz Z, Senturk S Characterization of lipid profile in dog with parvoviral enteritis. *J Small Anim Pract*. 2007;48(11):643–650.

25. SY Marulappa, Kapil. Simple test for rapid detection of canine parvovirus antigen and Canine parvovirus specific antibodies. *Clinical and Vaccine Immunology*. 2009;16:127–131.

26. Prittie J. Canine Parvoviral Enteritis; a review of diagnosis, management and prevention. *Journal of Veterinary Emergence Critical care*. 2004;14(3):167–176.

27. Veir JK. Canine parvoviral enteritis. In: Bonagura JD, Twedt DC, editor. *Kirk current veterinary therapy XV 15th edn*. USA: St Louis; 2014. p. 533–536.

28. Pollock RVH. The parvoviruses Part 11, Canine Parvoviruses Compend continue Education. *Pract Vet*. 1984;653–664.

29. Otto CM, Rieser TM, Brooks MB, et al. Evidence of hyper coagulatibility in dog with Parvoviral enteritis. *Journal of America veterinary medicine association*. 2000;217(10):1500–1504.

30. Kalli I, Leontides LS, Mylonakis ME, et al. Factors affecting the occurrence ,duration of hospitalization and outcome in Canine Parvovirus infection. *Res Vet Sci*. 2010;89(2):174–178.

31. Otto CM, Drobatz KJ, Soter C. Endotoxemia and tumour necrosis factor activity indog with naturally occuring Parvoviral Enteritis. *J Vet Intern Med*. 1997;11(2):65–70.

32. Macintire DK. Bacterial translocation, clinical implication and prevention In: Bonagure JD, editor. *Kirk current Veterinary therapy XIII Small Animal Practice*. 13th edn. Philadelphia: Saunders; 2000:201–203.

33. Hueffer K, Parrish CR. Parvovirus host range cell tropism and evolution. *Curr Opin Microbiol*. 2003;6(4):392–398.

34. RH Johnson, JR Smith. Epidemiology and pathogenesis of Canine Parvovirus Australia. *Veterinary Practice*. 1983;13:1.

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